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AROUSAL AND TASK PERFORMANCE: THE UBIQUITOUS U-CURVE,(U)
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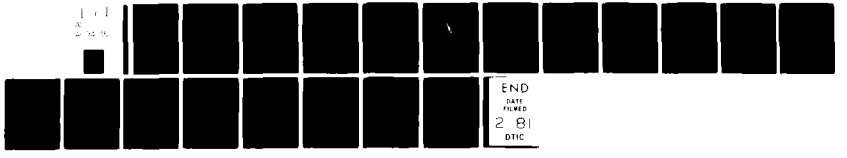

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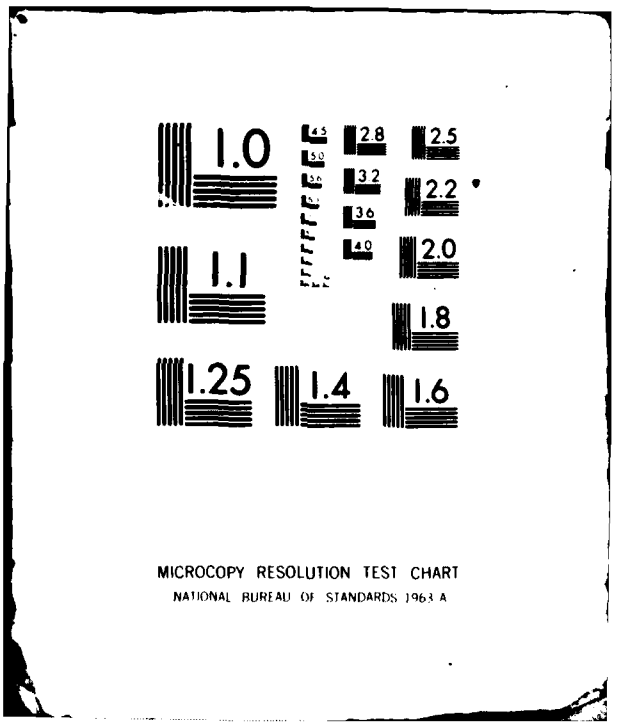
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SYSTEMS NOTE 68

AROUSAL AND TASK PERFORMANCE: THE UBIQUITOUS U-CURVE

by

K. C. HENDY

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(10) K. C. HENDY

SUMMARY

It is suggested that arousal is a combination of several relatively independent, imperfectly coupled mechanisms. This view is supported by the extensive literature on the dissociation of the electrocortical, autonomic and behavioural aspects of the arousal paradigm. In the case of electrocortical arousal an inverted U-curve can be demonstrated for tasks involving competing mechanisms (e.g. correct detection/false positives). The nature of this relationship is that arousal level (total) for optimum performance is greater for more difficult discriminations. This apparent contradiction of the Yerkes-Dodson Law *is explained in terms of the fear-induced drive peculiar to the experimental situation causing a high level of internal task-related arousal. Thus, measured incentive levels, which provide additional arousal through manipulation of the payoff matrix, may be seen to decrease as discriminations become more difficult.*

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16. *ABSTRACT*

It is suggested that arousal is a combination of several relatively independent, imperfectly coupled mechanisms. This view is supported by the extensive literature on the dissociation of the electrocortical, autonomic and behavioural aspects of the arousal paradigm. In the case of electrocortical arousal an inverted U-curve can be demonstrated for tasks involving competing mechanisms (e.g. correct detection/false positives). The nature of this relationship is that arousal level (total) for optimum performance is greater for more difficult discriminations. This apparent contradiction of the Yerkes-Dodson Law *is explained in terms of the fear-induced drive peculiar to the experimental situation causing a high level of internal task-related arousal. Thus, measured incentive levels, which provide additional arousal through manipulation of the payoff matrix, may be seen to decrease as discriminations become more difficult.*

CONTENTS

1. INTRODUCTION

Contemporary military systems are complex combinations of human and machine components. In many of these systems rapid technological advances have greatly increased machine capability and reliability to such an extent that the role of the human element in the system is often the critical determinant of overall performance. For example, a major proportion of aircraft accidents is attributed to human error rather than equipment failures *per se*. In 1977/78 this proportion was 75% for the RAAF (Lee (1978)). However, many human error accidents can be shown to be due to man-machine mismatches and may therefore have been avoided if an appropriate behavioural model, for the human operator, had been used in the system design.

It seems reasonable to reject the most basic models of behaviour that would have the stimulus-response link uniquely and rigidly formed. It is widely recognised that this relationship is mediated by various states inherent to the responding organism. The justification for this statement comes from the extensive literature on concepts such as attention, drive, motivation, set, expectancy, emotion, activation, arousal etc. These factors all refer to some assumed state of responsiveness of the organism (Broadbent (1971), Berlyne (1960) and Welford (1976) reference much of this literature). Duffy (1957) argued that all variations in behaviour could be explained by modification of the *direction of behaviour* and the *intensity of behaviour*. Within this context *attention* would be directional while *drive, motivation, arousal, emotion* are probably best thought of as mediators of intensity. *Set* and *expectancy* could be seen as mediating both direction and intensity of response.

Arousal and *activation* as the basis for theories of behaviour appear to stem largely from the observed physiological correlates of inferred behavioural states (e.g. Duffy (1957)). In EEG studies various distinguishable *arousal patterns* were associated with sleep, resting wakefulness, attentive behaviour, heightened emotional states etc. (Berlyne (1960)). These observations together with the growing knowledge of the role of the brain stem reticular formation in providing diffuse cortical innervation led Lindsley (1951) to propose his *Activation Theory of Emotion*. Despite the title of the theory and the context in which the argument was reported, it is evident that Lindsley intended the theory to be more broadly based. Lindsley states (p. 504):

> "... The activation theory is not solely an explanatory concept for emotional behaviour but relates also to the phenomena of sleep-wakefulness, to EEG manifestations of cortical activity, and to certain types of abnormal behaviour revealed in neurologic and psychiatric syndromes."

Although the terms *arousal* and *activation* are often used interchangeably (e.g. Duffy (1957)) there are soundly based reasons (Malmo (1957)) for preferring the more specific use of these concepts as follows:

(i) Activation; a state of physiological adaptation of the organism as manifested in the various physiological measures of cortical excitability (EEG and derived indices) and autonomic system state (GSR, heart rate, respiration rate etc.),

(ii) Arousal; the psychological responsiveness of the organism; the state that determines the intensity of the response.

To assume a conformal relationship between arousal and activation as defined here ignores the often tenuous relationship existing between them (Mehrabian and Russell (1974), Broadbent (1971)). Despite the apparent distinction just made between the two concepts, the circularity of the argument was well recognised by Malmo (1957) in stating the requirement for an objective measure in the behavioural dimension. However, to assume that the concepts are linked causally is fundamentally different from assuming they are linked conformally.

2. ACTIVATION AS A THEORY FOR BEHAVIOUR

Although Lindsley's formulation (*op. cit.*) could rightly be called an activation theory within the construct previously cited, the existence of such a mechanism was foreshadowed by the

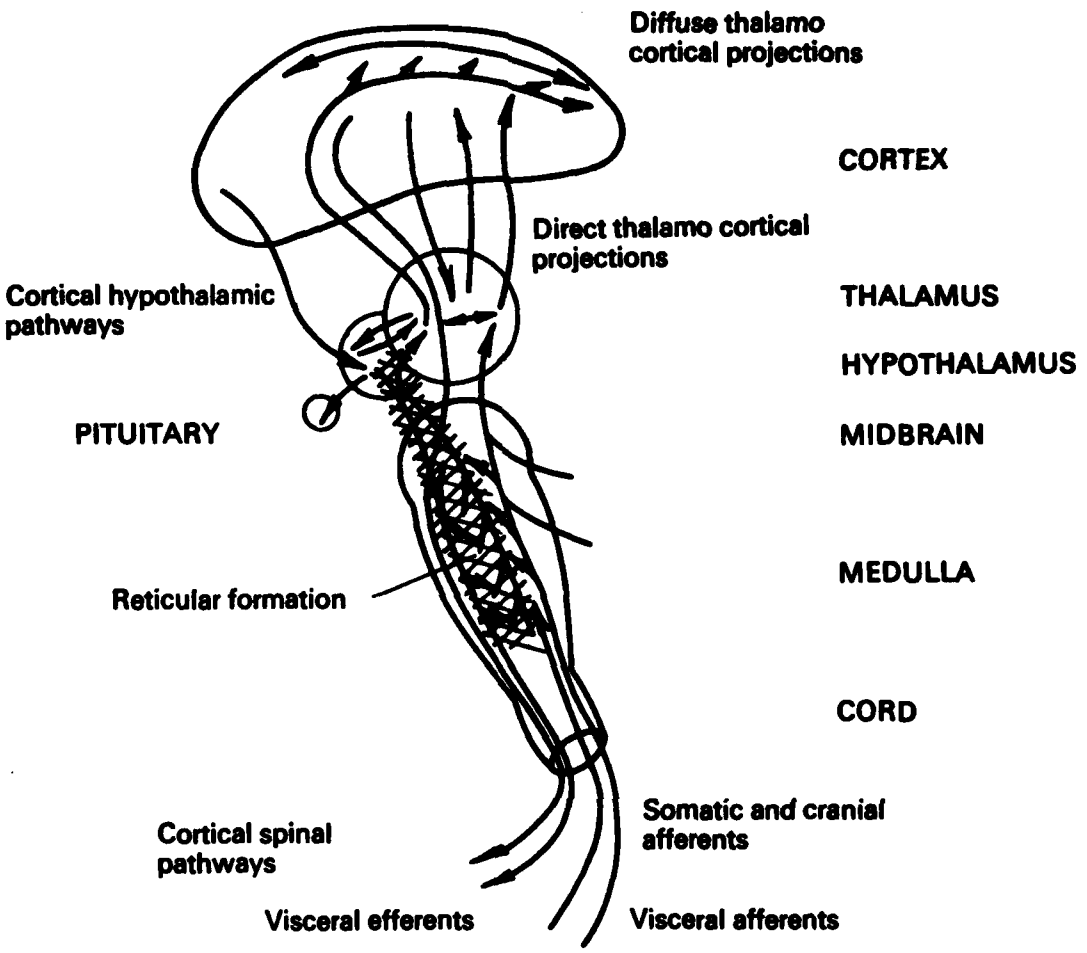

FIG. 1: SCHEMATIC REPRESENTATION OF THE ARAS

results of behavioural studies (Malmo (1959)). The evidence that Lindsley cites is largely physiological. At the center of the argument is the unravelling of the roles of certain brain stem regions (the thalamic region and reticular formation) in modifying the state of cortical activation (particularly EEG desynchronisation) and in producing overt behavioural responses in animal preparations. A schematic diagram of the ascending reticular activating system (ARAS—from Lindsley (1951)) is shown in Figure 1. The interconnections shown in this schema were largely supported by anatomical and physiological data.

Lindsley vested the activation properties of his model entirely in the reticular system. The evidence gathered at that time showed that direct stimulation of the reticular formation produced the *activation* pattern in the EEG (desynchronisation, low voltage high frequency activity) and selective destruction of this region produced a synchronised EEG pattern showing similar characteristics to the sleep spindles of normal (stage 1) sleep. The behavioural aspects were considered as follows (Lindsley (1951), p. 505):

> "... The mechanism of the basal diencephalon and lower brain stem reticular formation, which discharges to motor outflows and causes the objective features of emotional expression is either identical with or overlaps the EEG activating mechanism, ..., which arouses the cortex."

Thus Lindsley left open the possibility that behavioural arousal and electrocortical activity, although closely linked, may arise from different processes. This distinction seems to be lost in later work; for example Malmo (1959) in stating his version of the theory said:

> "... the continuum extending from deep sleep at the low activation end to *excited states* at the high activation end is largely a function of cortical bombardment by the ARAS such that the greater the cortical bombardment the higher the activation."

The activation part of the argument seems to be soundly based, as it draws on the strong physiological evidence relating to the function of the ARAS in determining cortical excitability. Indeed measures of cortical activity (spike frequency, EEG indices etc.) are usually continuous variables that lend themselves to ready expression on a continuum of measurement. Perhaps it is also meaningful to discuss a psychological continuum as suggested by Duffy (1962) on which can be plotted all states from somnolence through varying degrees of alertness to agitated , hyperactive states. The failure of activation theories to predict behavioural responses in a consistent and reliable fashion (Broadbent (1971), Stroh (1971)) may well lie in assumptions about the nature of the coupling between the two systems.

3. THE UNIFYING CONCEPT OF AROUSAL

Introspection and observation would suggest that task performance is usually submaximal when asleep or when stressed to a state of panic. The usefulness of such a concept as a predictive model of human behaviour depends mainly on the ability to bridge these limiting states by some continuum. The implication is that at some state(s) along this continuum between the extremes of sleep and panic, performance is optimal. Therefore there is a U-shaped curve of performance versus psychological state along this dimension. The unifying concept used to link these ideas has been the construct of *arousal* as previously defined. However, without an arousal metric the theory is impotent.

Duffy (1957) attributes the roots of the arousal concept to Cannon's *energy mobilisation* during *emotion*; however, Duffy extends the process to describe the intensity aspect of all behaviour. It seems that the only justification for conceiving arousal as a unidimensional concept came from the type of reasoning epitomized in the writings of Duffy, i.e. that all modifications of behaviour were explicable in terms of direction or intensity, with arousal determining the intensity factor. In the absence of a physiological explanation of such a generalised process, this approach was quite reasonable. However, once the roles of the thalamic and reticular regions became more clearly defined, the existence of a single intensity-determining state of the organism should have been questioned. It would appear that the various interconnections between brainstem regions (both afferent and efferent) and the cortex would suggest a more complex, interactive control of behavioural intensity than the unidimensional concept would imply.

The attempt to maintain arousal as a fundamental state variable of the organism has been strongly challenged (Lacey (1967), Stroh (1971), Mehrabian and Russell (1974)). Feldman and Waller (1962) showed that electrocortical activation and behavioural arousal are mediated by

separate brain stem regions and hence are functionally separate mechanisms. They found that electrocortical activity depended on the reticular formation while the posterior hypothalamic region controls the behavioural state. By selective isolation and stimulation of brain stem regions, Feldman and Waller were able to demonstrate that these two effects could be induced independently. Scott (1966) cites evidence that leads him to conclude that "... sleep is not part of the activation continuum." Nakajima (1964) was able to produce states of hyperactivity and *delirium* by injection of small amounts of neural depressants into the reticular formation. Larger doses produced a comatose state without an intervening state of drowsiness or sleep.

To the dual aspects of arousal differentiated by Feldman and Waller, Lacey (1967) added a third component—autonomic arousal. Lacey summarised as follows:

> "... the evidence shows that electrocortical arousal, autonomic arousal and behavioural arousal may be considered to be different forms of arousal, each complex in itself ... and which in general occur simultaneously."

The implication drawn from Lacey's argument is that a system *state* can be described as a point (or region) on a multivariable continuum having at least the dimensions of electrocortical arousal, behavioural arousal and autonomic arousal. The behavioural manifestations described by Duffy (1962) would correspond to regions on this continuum but may not project congruently onto the major axes. It is significant that independently of the approach taken by Lacey many authors have agreed that there is no single physiological indicator of arousal (Duffy (1962), Stroh (1971), Spyker *et al.* (1971)). Such a finding at least is in harmony with the multidimensional approach.

4. A MULTIDIMENSIONAL APPROACH TO AROUSAL

Recognition that there is no single bodily mechanism determining the arousal state may prove to be beneficial in promoting a better understanding of the phenomena. Most studies appear to have assumed that the physiological parameters measured represent steady state measures. Taylor and Epstein (1967) suggest that the various systems responding to imposed stress are likely to vary in their temporal properties. Stroh (1971) also claims that in addition to the specific task-related responses there is a long term need to control the homeostasis of each subsystem.

Some of the paradoxes of arousal theory that concerned Broadbent (1971) may have their explanations vested in the type of multidimensional construct supported in this paper. Aspects of Lacey's three way split of arousal are discussed in the following sections.

4.1 Electrocortical Arousal

It is assumed that cortical tone is mediated by the diffuse projection system of the ARAS which is in turn innervated by collaterals of the various sensory systems. The diffuse activation of the cortex will depend on the total amount of sensory data passing to the reticular formation. This line of thinking is compatible with a signal detection approach to the processing of sensory data in line with Malmo's (1959) statement "... the greater the cortical bombardment the greater the activation." This type of stand was taken by Welford (1976), albeit from a unidimensional viewpoint.

To consider only the rising pathways from ARAS to the cortex implies that cortical activity depends only on sensory input. This situation seems to deny the ability of the organism to internally mediate cortical arousal. However, French *et al.* (1955) found that specific cortical areas project downwards to the brain stem reticular formation. Such descending pathways were also included in Lindsley's (1951) schema of the ARAS (Fig. 1). Lindsley (1961) and Murrell (1967 and 1969) proposed that these pathways provided a means of cortical influence on the arousal function. Murrell (1967) used the term *auto arousal* to refer to "... cortical activation resulting from stimulation of the reticular formation by the cortex, this stimulation being under voluntary control". Some type of internal mechanism is necessary to explain *motivation* and the assumed arousing effects of incentives, knowledge of results etc. The arousing properties of such constructs probably lie in cognition rather than in their direct sensory input.

It seems that no arousal theory is complete unless it can be demonstrated that the model implies an inverted U-shaped curve connecting performance to arousal (Broadbent (1965),

Welford (1976), Murrell (1967) and (1969), Berlyne (1960), Scott (1966) etc.). Several authors have attempted to postulate mechanisms by which performance might suffer at both low and high levels of cortical activity (Murrell (1967), Broadbent (1971), Welford (1962)). Welford (1976) used signal detection theory to demonstrate such a relationship at a cortical activation level.

4.2 Behavioural Arousal

The proponents of the unidimensional arousal theories have usually anchored their scales at the *sleep state*. In fact arousal as a unifying concept has been defined as:

"... *a measure of how wide awake the organism is* ..." (Berlyne (1960)) *or,*

"... the inverse of the probability of the subject falling asleep ..." (Corcoran (1963)).

However, the sleep-wakefulness state has a bistable character to it that could be thought of as overlying the smoothly varying electrocortical arousal function. Some studies of sleep deprivation (Ainsworth and Bishop (1971)) seem to support the view that performance may be maintained up to a point at which the subject *goes to sleep*. Performance in the sleep deprived state tends to be characterised by *errors of omission* (Welford (1976)).

It is assumed that sleep deprivation increases the pre-disposition to the sleeping state. Malmo and Surwillo (1960) found that the physiological measures of autonomic arousal (GSR, respiration rate, heart rate and muscle tension) and electrocortical activity (EEG) indicated a heightened state of activation during vigils lasting 60 hours. They cited the animal studies of Bunch *et al.* and Licklider and Bunch that showed that the performance of sleep deprived rats was superior to controls. In a 1946 study Licklider and Bunch conclude:

"... the better performance of the wakeful rats is largely to be accounted for in terms of their more aggressive behaviour."

and that restriction of the animals' sleep made them "... highly irritable and highly reactive".

These observations are not surprising, as Bunch and Licklider's rats must have been stimulated with some compelling sensory inputs in order to keep them awake. This input, driving the ARAS, should maintain electrocortical activity at least. In the case of Malmo and Surwillo's subjects, the authors claim they appeared to have a high intrinsic motivation (volunteers) and interest in the project. In this type of experiment it seems reasonable to assume that the pre-disposition to sleep is increasing as sleep deprivation increases; hence, some system state is changing in a direction that would increase the probability of sleep. This, despite cortical and autonomic indicants of activity showing an aroused state.

The effect of alcohol interacts synergistically with incentive (Wilkinson and Colquhoun (1968)) although alcohol is considered to be a cortical depressant. The interaction of alcohol with sleep deprivation gave equivocal results. Broadbent (1971) suggests that the evidence is that in small doses alcohol causes performance to change consistent with it acting as an arouser. In large doses Wilkinson and Colquhoun found that alcohol increased the effect of sleep loss. Perhaps these findings just demonstrate a differential action of alcohol on the ARAS and thalamo-cortical systems.

It seems that the type of result found in these *multi-stressor* experiments is more consistent with a sleep-wakefulness system overlying and acting relatively independently of the autonomic and cortical arousal systems. The nature of this system is not clear and is obviously complex (Mountcastle (1974)), particularly in the sleeping state. Mountcastle offers physiological evidence that appears to be consistent with this view. Although recognising the tentative nature of the hypothesis he suggests that there is strong evidence to support the view that in addition to the ARAS there is an antagonistic group of sleep inducing structures in the lower brain stem. Both these systems may act on the thalamic pacemaker with the final outcome depending on the relative degree of activity in each.

4.3 Autonomic Arousal

The autonomic nervous system (ANS) consists of an efferent outflow to the visceral organs, providing a system which adjusts body states and supports somatic reactions (Koizumi and Brooks (1974)). The two divisions of the ANS (sympathetic and parasympathetic) terminate on a variety of organs including the ciliary muscles, sphincter muscle of the iris, hair, sweat glands,

heart, liver, intenstine, bladder etc. Stimulation of the ANS produces a number of changes that are said to prepare the organism for *fight or flight*, for example, pupil constriction, increased cardiac output, reduced blood volume in the viscera, decreased blood clotting time, sweating, hypertension etc.

Much of the ANS is tonically active, hence, visceral organs are normally held in an intermediate state. Thus visceral outputs may change in sympathy with a diminution or augmentation in the rate of efferent fibre firing. Visceral organs may be jointly innervated by both sympathetic and parasympathetic fibres which may have inhibitory or excitatory effects and act either synergistically or antagonistically (Koizumi and Brooks, *op. cit.*). Hence, there is the potential for a large repertoire of visceral responses to ANS activity.

Broadbent (1971) in referring to the inconsistent response of peripheral physiological measures (visceral organ responses) under the arousal paradigm, suggests that these measures may reflect at least two states, *arousal* and *effort*. Such a stand is appropriate to the historically held concept of the ANS acting in a supportive role "... to prepare the organism, realistically or unrealistically, for vigorous goal directed activity requiring the mobilization and expenditure of energy and to protect the organism against the hazards of such vigorous activity" (Scott (1966)). Both this assumed role and the diverse functions performed by the host of visceral organs served by the ANS would suggest that the pattern of autonomic responses would be highly task dependent. Hence, it would be expected that the autonomic arousing response to a task that imposed a severe threat to life and limb would be different from the response to a task that required fine manipulation but posed no physical threat. In other words it is suggested that the set of autonomic measures is a subset of the system variables that completely describe a given state. The specificity of the ANS is described succinctly by Koizumi and Brooks (p. 790) with respect to the parasympathetic division:

> "... It is obvious that there could be no physiologic rationale for the discharge of the parasympathetic system as a whole. Simultaneous dilation of the pupil, salivation, slowing of the heart, increased activity of the gut, defecation, urination and erection of the penis have no sensible functional association. These phenomena are associated only in abnormal circumstances that produce a mass reflex. The components of the parasympathetic system behave independently, participating in specific reflexes or well integrated reactions."

Whereas arguments can be advanced that suggest how performance may depend on electrocortical activity and behavioural arousal (sleep-wakefulness), the interaction between autonomic arousal and performance is probably a second order effect, e.g. through the secretion of substances into the blood stream that have a facilitatory effect on the ARAS or cortex (Berlyne (1960)). Hence, the ability of ANS measures to predict performance changes depends on the relationship between ANS functioning and the mechanisms that are responsible for the performance decrement. It is this relationship that is usually assumed to be monotonic, with little justification in view of the sometimes competing nature of the two divisions of the ANS.

5. PERFORMANCE AND CORTICAL AROUSAL

As stated previously the classical relationship between efficiency of performance and arousal level is assumed to be in the form of an inverted U. Such a relationship is to be expected if competing mechanisms are involved (Malmo (1959)). Malmo uses the concept of *habit interference* as the competing phenomenon. He suggests that the inverted U-curve would be expected to show most strongly in complex tasks where final outcome depended on the relative strengths of the two mechanisms. For simple tasks, therefore, in which habit interference effects are negligible, the relationship between performance and arousal would be monotonic. However, Malmo summarises experimental evidence that suggests that even simple tasks (bar pressing, salivation response) are characterised by a non-monotonic relationship.

Such generalised concepts as provided by Malmo's (1959) habit interference hypothesis do not provide a mechanism to explain why arousal and performance interact in any particular fashion. If the single aspect of electrocortical arousal is considered, however, the application of a signal detection approach yields a relationship between performance and activation that does suggest an underlying neural explanation (Welford (1976)).

5.1 The Signal Detection Model

The rationale behind the signal detection approach to cortical processes is covered elsewhere in considerable detail (Welford (1976), Broadbent (1971) and (1965) reference much of this work). For the purposes of this paper it is sufficient to summarise the basic assumptions of Welford's (1976) thesis as follows. Suppose the diffuse reticulo-cortical projections act in a facilitatory way. Hence, increased activity in the ARAS would increase the general level of cortical activity both in the presence of a signal and in the no-signal case. Thus, arousal acts in a multiplicative fashion, expanding noise and signal plus noise distributions upwards on the activation axis, while leaving the judgemental criteria fixed.

Welford cites a simple signal detection task to illustrate his point. Suppose that a subject must judge whether a signal has been presented on a number of trials, under varying levels of arousal (as manifested in the amount of cortical activity). The probabilities of *signal* and *no signal* for each trial are equal and a relationship between proportion of correct responses and activation level is required. Hence, if:

$$P(C) = \text{probability of a correct response,}$$

$$P(sn) = \text{probability of a signal,}$$

$$P(n) = \text{probability of no signal,}$$

$$P(Y/sn) = \text{probability of a correct detection,}$$

$$P(N/sn) = \text{probability of an incorrect rejection,}$$

$$P(N/n) = \text{probability of a correct rejection,}$$

$$P(Y/n) = \text{probability of an incorrect detection, then}$$

$$P(C) = P(Y/sn).\ P(sn) + P(N/n).\ P(n).$$

When $P(sn) = P(n) = 0{\cdot}5,$

$$P(C) = 0{\cdot}5\,\{P(Y/sn) + P(N/n)\}. \tag{1}$$

This computation should be made as the distributions are expanded from being wholly to the left of the criterion point to the opposite extreme (the word *wholly* is used in a conceptual sense, as the distributions used to model the process may have infinite tails). The amount of expansion is a measure of the arousal level.

The nature of the computations used by Welford to demonstrate an inverted-U relationship is not specified (Welford (1973) and (1976)), although they are presumably based on the type of argument used here. Although the general form of the performance curve presented by Welford is not in dispute, the finer details of the derivation are at issue, particularly with regard to the fitting of the classical data of Yerkes and Dodson (1908).

To support this objection to Welford's conclusions, Equation (1) was computed for three levels of signal discriminability (i.e. a d' of 0·5, 1·0 and 1·75). The assumed noise-alone distribution and the three signal plus noise distributions are shown in Figure 2. For computational simplicity a *Normal* form (with equal variances) has been used. The use of the *Normal* form has considerable justification (Eijkman and Vendrik (1963)) and does not change the nature of the conclusions to be drawn.

As a further aid to computation, rather than rigorously expand the scale of the distributions against a fixed criterion, an equivalent process is used; i.e. causing the criterion level to sweep across the distributions (kept fixed) by an appropriate rescaling. The magnitude of this rescaling is taken as the measure of arousal level. Figure 3 shows the result of sweeping a variable criterion across the fixed distributions of Figure 2. The variable criterion (c) is moved from left to right (decreasing arousal) across the arbitrary and normalised scale of neural activity. Note that the scale of neural activity in Figure 2 has been normalised to give unit standard deviation and zero mean for the noise-alone distribution.

However, the abscissa of Figure 3 does not represent a scale of arousal. As stated previously, arousal should be measured by the amount of expansion of the distributions of noise and signal plus noise (or equivalently by the contraction of the criterion level). Thus if the abscissa of

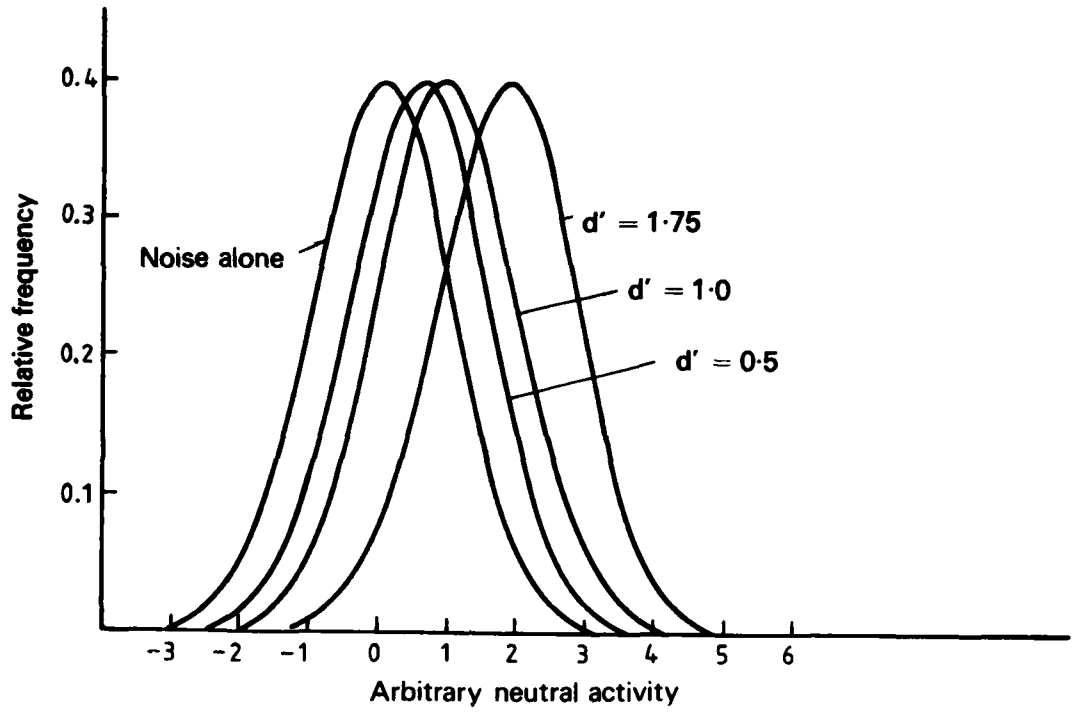

FIG. 2: NOISE AND SIGNAL PLUS NOISE DISTRIBUTIONS

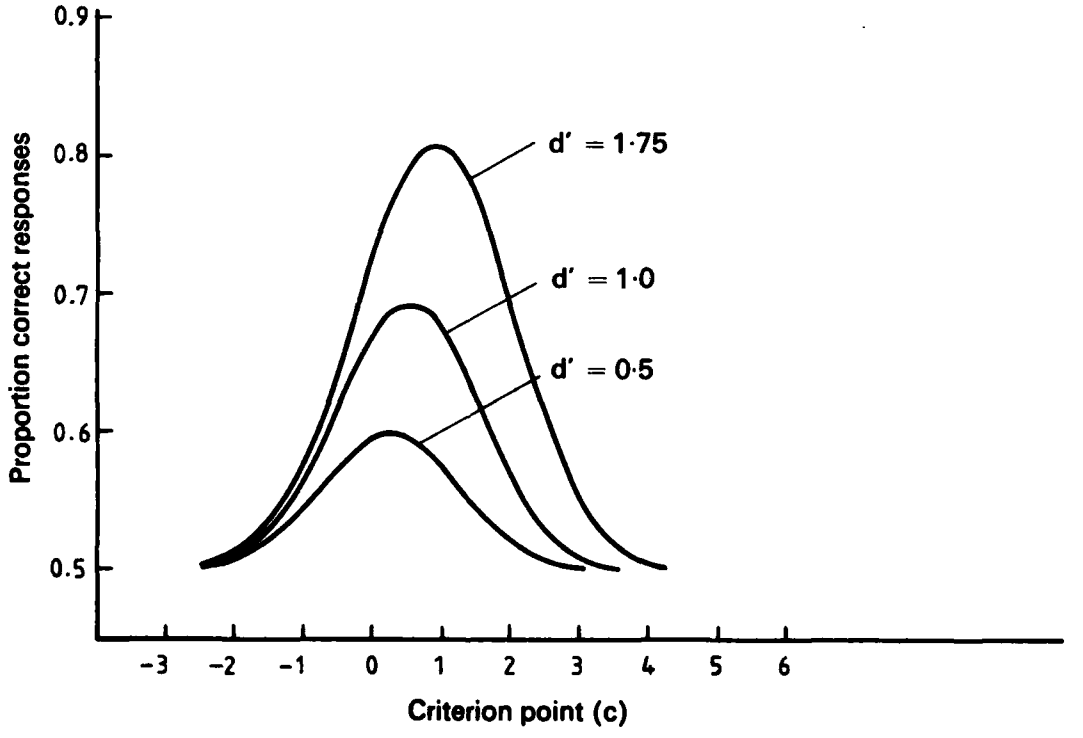

FIG. 3: PROPORTION OF CORRECT RESPONSES AS A FUNCTION OF CRITERION POSITION

Figure 3 is a measure of the moving criterion level (c) against the rescaled distributions, then the rescaling factor (k) is a measure of arousal, where:

$$k = \frac{(4{\cdot}25\text{-}c)}{6{\cdot}75} \quad (0 \leqslant k \leqslant 1);$$

k has been arbitrarily scaled to have values of 1·0 at $c = -2{\cdot}5$ and 0·0 at $c = +4{\cdot}25$. This arbitrary fixing of the k-scale can be interpreted as implying 100% arousal when the two distributions lie predominantly to the right of the criterion point (at $c = -1{\cdot}25$) and 0% arousal when mainly to the left (at $c = +4{\cdot}25$). Proportion of correct responses against the arousal factor k is shown in Figure 4 (computations are given in Table 1).

Figure 4 demonstrates several important points, viz.:

(i) as d' decreases $P(C)_{max}$ also decreases;

(ii) as d' decreases the level of arousal for $P(C)_{max}$ moves to the right (c.f. with Welford (1976), Figure 7·3); and

(iii) the three U-curves converge at the right hand end of the scale (c.f. also with Welford's Figure 7.3).

TABLE 1
Computation of the Proportion of Correct Responses

c	k	$P(C)$		
		$d' = 0{\cdot}5$	$d' = 1{\cdot}0$	$d' = 1{\cdot}75$
−2·5	1·000	0·502	0·503	0·503
−2·25	0·963	0·505	0·506	0·506
−2·00	0·926	0·508	0·511	0·511
−1·75	0·889	0·514	0·519	0·520
−1·50	0·852	0·522	0·530	0·533
−1·25	0·815	0·533	0·547	0·552
−1·00	0·778	0·546	0·568	0·578
−0·75	0·741	0·561	0·593	0·610
−0·50	0·704	0·575	0·621	0·648
−0·25	0·667	0·587	0·648	0·689
0·00	0·630	0·596	0·671	0·730
0·25	0·593	0·599	0·686	0·766
0·50	0·556	0·596	0·692	0·793
0·75	0·519	0·587	0·686	0·807
1·00	0·481	0·575	0·671	0·807
1·25	0·444	0·561	0·648	0·793
1·50	0·407	0·546	0·621	0·766
1·75	0·370	0·533	0·593	0·730
2·00	0·333	0·522	0·568	0·689
2·25	0·296	0·514	0·547	0·648
2·50	0·259	0·508	0·530	0·610
2·75	0·222	0·505	0·519	0·578
3·00	0·185	0·502	0·511	0·552
3·25	0·148		0·506	0·533
3·50	0·111		0·503	0·520
3·75	0·074			0·511
4·00	0·037			0·506
4·25	0·000			0·503

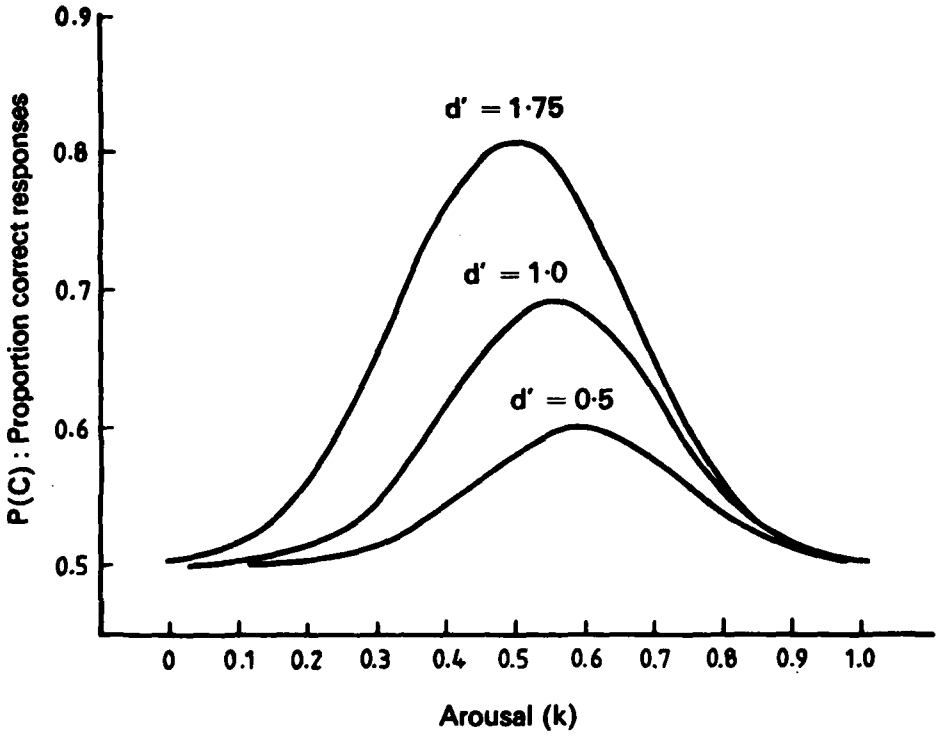

FIG. 4: PERFORMANCE VERSUS AROUSAL

In the light of the contradictions of Welford's conclusions, points (ii) and (iii) require further discussion. Consider the form of the performance curves. As arousal increases and the distributions of neural activity expand against the fixed criteria, the distributions progress from being generally to the left of the cut-off to being generally to the right of the cut-off. Hence, as arousal increases, the first change in *number of correct responses* must come mainly from the increase in $P(Y/sn)$ as the *sn* distribution expands across the criterion point. The amount of expansion required for $P(Y/sn)$ to start to increase will be determined by the relative position of the *sn* distribution on the activity axis. As the *n* distribution is assumed to be independent of the signal, the position of the *sn* distribution depends on d'. Thus the curves of $P(C)$ against arousal should separate at the low arousal end as shown in Figure 4. Similarly at the high arousal end, performance depends ultimately on the increase of $P(Y/n)$, which in turn depends on the noise-alone distribution. Thus the curves of $P(C)$ versus arousal should converge at the high arousal end.

Perhaps the most significant departure of the present results from those of Welford is the movement of the performance peak as discrimination becomes more difficult. The present results indicate that a higher level of arousal is necessary for optimum performance at a difficult discrimination task than for an easy one, in direct opposition to the Yerkes-Dodson Law (Welford (1976)). But it seems the explanation for this apparent paradox lies in the interpretation of the Yerkes-Dodson experiments and the type of generalisations made from these data to other loosely related situations.

5.2 The Yerkes-Dodson Law

Welford (1968) briefly reviews the original experiments conducted by Yerkes and Dodson. In these experiments mice had to learn to discriminate between two different brightnesses, receiving an electric shock for a wrong decision. For an easy discrimination the number of trials to learn the correct response decreased as the strength of shock increased. For difficult discriminations the number of trials also fell as shock intensity increased, but a level was reached after which number of trials began to rise. For difficult discriminations the optimum shock was weaker than for those of moderate difficulty. Welford further references a number of studies using different tasks and incentives, involving both animal and human subjects, which have shown similar outcomes. He offers two explanations for these results. Firstly, that strength of shock is equivalent to strength of incentive (a motivating concept) and that neural activity is proportional to incentive. In this case the Yerkes-Dodson results would not be explained by the present findings. Secondly, that the tasks have an inherent arousing value depending on their *degree of difficulty*. The task induced factor would add to that due to the incentive, to determine the overall level of cortical activity. This second explanation is favoured, as agreeing more closely with the experimental evidence.

Apart from the findings of Yerkes and Dodson (in their specific experimental situation) there seems to be no reason for expecting that difficult discriminations are best performed at low levels of arousal. On the contrary, those states typically described as being low in arousal (drowsiness) are just the states where small signals are likely to be missed while larger ones may be detected (see Stroh (1971) for the effects of signal strengths in offsetting vigilance decrements). If the discrimination is difficult we may have to *pay attention, make up our mind to do well* (Murrell (1967)), *try harder* etc. These are the types of concepts discussed by Murrell in terms of auto-arousal, and indeed seem to describe a conscious effort by the organism to modify some internal state, in order to improve performance.

It is suggested that the overall activation level is made up of three components, viz.:

(i) a task-independent aspect (personality (Corcoran (1965)), psychiatric conditions (Malmo (1957)) etc.);

(ii) a task-dependent aspect which depends on the individual's payoff matrix; and

(iii) a task-specific factor due directly to the stimulation of the organism by the incoming sensory information.

The key to the interpretation of the Yerkes-Dodson experiments may lie in the second factor. The payoff matrix is nothing more than the organism's perceived rewards and costs for the various experimental outcomes (Egan (1975)). Note that in determining the weights of the payoff matrix it is the perceived value of monetary rewards, costs, punishments (the utility) that

is important. In some cases the perceived value may not be linearly or even monotonically related to the external stimulus (e.g. adaptation to cutaneous pain and thermal stimulation (Jenkins (1951)). In the Yerkes-Dodson experiments large shock intensities imply a high cost of making an incorrect decision. Miller (1951) cites examples of how such threatful situations can invoke fear responses, avoidance behaviour and autonomic arousal responses even in the absence of the punishing stimulus; that is, these anxiety producing situations have an anticipatory arousing property.

The Yerkes-Dodson experiments are essentially a learning situation with fear being the learned drive (Miller (1951)). It seems critical to the argument that in order to learn to make the correct discrimination, first the subject must perceive that there is a difference in the stimuli and correctly identify the direction of this difference. Further the subject must pair the reward (lack of shock) with the correct discrimination for reinforcement to be effective. Thus in many cases the reward will be coupled with an incorrect perception or even coupled with extraneous cues, thereby slowing the learning process and reducing the opportunity for drive reduction through success (Miller (1951)). When the stimuli become indiscriminable, success is a chance occurrence, as is punishment, with no way open for the organism to influence this anxiety producing situation. This is presumably the limiting case in which the internal fear-induced component of arousal is greatest.

Thus the situation is seen in which more difficult discriminations, together with a strong drive inducing payoff matrix, result in an increasing fear/anxiety induced arousal component. Hence, if the rate of increase in this internal component is greater than the upways shift of the optimum $P(C)$, then externally added arousers (shock strength for example) would be seen to decrease for optimal performance as discriminations became more difficult. This is the Yerkes-Dodson result.

6. CONCLUSIONS

It is suggested that arousal is a combination of several relatively independent, imperfectly coupled mechanisms. This view is supported by the extensive literature on the dissociation of the electrocortical, autonomic and behavioural aspects of the arousal paradigm. In the case of electrocortical arousal an inverted U-curve can be demonstrated for tasks involving competing mechanisms (e.g. correct detection/false positives). The nature of this relationship is that arousal level (total) for optimum performance is greater for more difficult discriminations. This apparent contradiction of the *Yerkes-Dodson Law* is explained in terms of the fear-induced drive, peculiar to the experimental situation, causing a high level of internal task-related arousal. Thus the measured incentive levels (which provide additional arousal through the manipulation of the subject's payoff matrix) for optimum performance may be seen to decrease as discriminations become more difficult. Such a situation would typically arise in an experiment in which there was a high utility associated with making a correct response (or alternatively in not making an incorrect response). In this present paper the possible effects on the criterion level of manipulating the payoff matrix have not been considered.

When the performance measure does not involve competing mechanisms the relationship between performance and arousal might be expected to be single valued. Simple reaction time (for correct detections) is a performance measure that may show such a relationship. Hence, although the proportion of correct responses may decrease at the upper end of the cortical excitability scale, there appears to be no reason to suspect that reaction time would suffer a corresponding degradation.

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